# REPORT DOCUMENTATION PAGE

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| 1a REPORT SECURITY CLASSIFICATION<br>UNCLASSIFIED | 1b RESTRICTIVE MARKINGS |
|---|---|
| 2a SECURITY CLASSIFICATION AUTHORITY | 3 DISTRIBUTION / AVAILABILITY OF REPORT<br>Approved for public release; distribution unlimited. |
| 2b DECLASSIFICATION / DOWNGRADING SCHEDULE | |
| 4 PERFORMING ORGANIZATION REPORT NUMBER(S) | 5. MONITORING ORGANIZATION REPORT NUMBER(S)<br>AFOSR·TR· 87-1681 |

| 6a NAME OF PERFORMING ORGANIZATION | 6b OFFICE SYMBOL<br>(If applicable) | 7a. NAME OF MONITORING ORGANIZATION<br>Air Force Office of Scientific Research/NL |
|---|---|---|
| 6c ADDRESS (City, State, and ZIP Code) | | 7b ADDRESS (City, State, and ZIP Code)<br>Building 410<br>Bolling AFB, DC   20332-6448 |

| 8a. NAME OF FUNDING / SPONSORING<br>ORGANIZATION<br>AFOSR | 8b OFFICE SYMBOL<br>(If applicable)<br>NL | 9. PROCUREMENT INSTRUMENT IDENTIFICATION NUMBER<br>AFOSR-86-0182 |
|---|---|---|

| 8c ADDRESS (City, State, and ZIP Code)<br>Building 410<br>Bolling AFB, DC   20332-6448 | 10 SOURCE OF FUNDING NUMBERS | | | |
|---|---|---|---|---|
| | PROGRAM<br>ELEMENT NO.<br>61102F | PROJECT<br>NO. | TASK<br>NO. | WORK UNIT<br>ACCESSION NO |

11 TITLE (Include Security Classification)

12 PERSONAL AUTHOR(S)

| 13a TYPE OF REPORT<br>Reprint | 13b TIME COVERED<br>FROM          TO | 14. DATE OF REPORT (Year, Month, Day) | 15. PAGE COUNT |
|---|---|---|---|

16 SUPPLEMENTARY NOTATION

| 17 | COSATI CODES | | 18 SUBJECT TERMS (Continue on reverse if necessary and identify by block number) |
|---|---|---|---|
| FIELD | GROUP | SUB-GROUP | |

19 AB                    on reverse if necessary and identify by block number)

AD-A187 697

DTIC
SELECTED
NOV 3 0 1987
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| 20. DISTRIBUTION / AVAILABILITY OF ABSTRACT<br>☒ UNCLASSIFIED/UNLIMITED  ☒ SAME AS RPT  ☐ DTIC USERS | 21. ABSTRACT SECURITY CLASSIFICATION<br>UNCLASSIFIED |
|---|---|
| 22a. NAME OF RESPONSIBLE INDIVIDUAL<br>Dr. William Berry | 22b. TELEPHONE (Include Area Code)<br>(202) 767-5021 | 22c. OFFICE SYMBOL<br>NL |

87 11 17 037

Two Attentional Models of Classical Conditioning:
Variations in CS Effectiveness Revisited

Nestor A. Schmajuk and John W. Moore

COINS Technical Report 87-29
April 3, 1987

# Computer and Information Science

## University of Massachusetts at Amherst

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# PREFACE

Connectionist approaches to cognitive performance are generally based on traditional network or associative frameworks in which learning plays a pivotal role. Studies of learning within these frameworks have long been a staple of those areas of experimental psychology concerned with cognitive performance and its ontology. Kamin's rediscovery of blocking of classical (Pavlovian) conditioning in rats twenty years ago triggered a movement within animal learning psychology that stresses the informational and cognitive aspects of tasks such as conditioning, discrimination learning, and stimulus generalization. Theoretical models that have grown up around these newer approaches have been extended to a variety of problems of computation and process control. Some of these extensions and applications have been described in recently published proceedings of the Cognitive Science Society and in papers generated by Professor A. G. Barto's Adaptive Network Group.

Many network learning algorithms conform to the delta rule. One important member of this class of algorithms is the Widrow-Hoff rule which Barto and his associates have shown to be closely related to an influential theory in psychology, the Rescorla-Wagner (RW) model. The RW model was devised as a mechanistic account of blocking and other multiple-cue protocols in the experimental literature on classical conditioning. The RW model and two theories described in this report can also be applied to a problems of interest to adaptive network researchers. One such problem area is that of credit assignment. Multiple-cue training protocols in classical conditioning have been cast in these terms, as Barto and Richard Sutton have illustrated in a series of reports. It has lately become commonplace to assess network learning algorithms by their ability to emulate the phenomenology of classical conditioning.

Useful as the RW model has proven to be, some of its competitors in the animal learning literature seem equally capable, if not more so. This report describes simulation studies of two of these alternatives to the RW model within the framework of classical conditioning. The results of these simulation studies suggest alternative computational forms of both models. These revised models have been successfully applied to neural network theories of hippocampal function and the formation of spatio-temporal cognitive maps.

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# Two Attentional Models of Classical Conditioning:
## Variations in CS Effectiveness Revisited

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### Abstract

Attentional models offer alternatives to the highly successful theory of Rescorla and Wagner (1972) for describing blocking, overshadowing, and many other features of classical conditioning. Two such models are the Moore and Stickney (1980) version of Mackintosh's (1975) attention theory and the Pearce and Hall (1980) model. These models emphasize variations in the associability of CSs instead of variation in the effectiveness of the reinforcing event, the US. Early published variants of the Moore-Stickney and Pearce-Hall models do not always accurately portray the effects of nonreinforced CS presentations as represented in simulation experiments. In the case of the Moore-Stickney model, levels of conditioned responding under partial reinforcement are too low to reasonably approximate expectations based on the experimental literature, and extinction is too deep to produce the rapid reacquisition that typically follows extinction. These problems are corrected by changing the expressions in the model for decreasing associative strength. The revised model retains the positive features of the original, e.g., the ability to simulate in real-time latent inhibition and compound CS effects such as blocking and conditioned inhibition. The P-H model is path dependent and highly nonlinear under partial reinforcement. The problem can be corrected either by modifying and restricting the rules for computing the associability of the CS, or by modifying the rules for computing associative strength. The revised model retains the original's ability to simulate latent inhibition, compound CS effects, and the transfer (positive or negative) from training with a weak US to training with stronger US.

## Introduction

This article reviews two mathematical models of classical conditioning that stress attentional processes, the Moore-Stickney (M-S) model (Moore & Stickney, 1980, 1982, 1985) and the Pearce-Hall (P-H) model (Pearce & Hall, 1980). These models feature mechanisms for altering the contribution of the CS to the rate of change of the associative relationship between a conditioned stimulus (CS) and the unconditioned stimulus (US), and they have been dubbed "attentional" models by their originators. Thus, instead of citing variations in the effectiveness of the US to account for the phenomena of conditioning as does,

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e.g., the Rescorla-Wagner (R-W) model, these models emphasize processes that cause the associability of the CS to vary. In assessing the M-S and P-H models in simulation experiments, we discovered some serious shortcomings in published versions of each. In this article we review the mathematical statement of both models in detail, shortcomings are indicated, and suggested modifications noted. These modifications are designed to enhance further development of each model into domains where computational versions of attentional theories of learning might find application, including neuroscience and artificial intelligence.

Following Rescorla and Wagner (1972), the symbol $V$ (for associative value) is used throughout to denote the primary theoretical dependent variable representing the strength and sign of the associative relationship between a CS and the US. Linear difference equations express how $V$ changes from one trial to the next. Although the resulting associative structures at the representation level are not isomorphic with performance measures such as the probability of a conditioned response (CR), the mapping from $V$ to behavioral indices of learning are at least monotonic in most applications (see Frey & Sears, 1978, for an extended treatment of this issue in relation to classical conditioning of the rabbit eyeblink).

Assumptions and formal structure of mathematical models of classical conditioning in the contemporary theoretical literature basically revolve around two questions. One question is whether the strength of associative links to the US among various components of a compound CS must be partitioned or shared (the "zero-sum" rule), as in the case of the R-W model, or whether a given component CS can in principle develop a complete associative link to the US despite the presence of competitors. Mackintosh (1975) has provided a lucid discussion of this point, and the distinction finds a parallel in the field of artificial or machine learning where the "zero-sum rule" is a feature of ADALINES and noncompeting associations is a feature of perceptrons (see Duda & Hart, 1973; Sutton & Barto, 1981).

The second question concerns the relationship between the rate of learning or, equivalently, the magnitude of the change in associative value on a given trial by a given CS, and the previous history of reinforcement of the CS. This question reduces to one of deciding whether the parameter of a given model that determines changes in $V$ of the CS remains invariant over training trials, as in the R-W model, or whether CS associability is permitted to change from one trial to the next. Some models, such as Mackintosh's (1975) attention theory and the M-S model, which do not feature a zero-sum rule, and Frey and Sear's (1978) "catastrophe" model, which does, assume that reinforcement momentarily increases the salience or associability of the CS and thereby its contribution to the rate of learning. Other models assume that the salience or associability of a CS decreases because of "reduced processing", as in Wagner's (1976) habituation theory and the P-H model.

Our interest in the M-S and P-H models arose from efforts to nurture a link between

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learning theory and the literature from neurobiology on the role of the hippocampus in learning and memory, particularly classical conditioning. The rationale for chosing attentional models for developing this linkage has been enunciated by the authors in other articles (Moore, 1979; Schmajuk, 1984; Schmajuk & Moore, 1985). We do not claim that other models cannot also provide the basis of a rigorous theoretical approach to the neurobiology of learning in its various forms. We simply do not feel qualified to proffer comment on all extant learning models. Any benefits in enhanced understanding of the hippocampus or the nature of classical conditioning accruing from these efforts must ultimately rest on how well our models perform in describing even the most mundane features of this type of learning.

## Overview

Using Mackintosh's (1975) attention theory as a scaffold, the M–S model uses a simple linear difference equation to express associative increments from one trial to the next during a simple acquisition protocol: When a trial is defined as the paired occurrence of the CS and US,

$$\Delta V = \alpha \theta (1 - V) \tag{1}$$

$\alpha$ is the rate parameter contributed by the CS, $\theta$ is the rate parameter contributed by the US, and $\alpha$ and $\theta$ are between 0 and 1. It has become customary to refer to $\alpha$ as the *associability* of the CS. The asymptotic level of learning equals 1 rather than the US-intensity dependent parameter $\lambda$ in Mackintosh's original paper.

Our reason for restricting the asymptotic level of associative strength to 1 is that we interpret $V$ to represent the strength of the organism's "belief" that the US follows the CS. Anthropomorphically, an organism can be no more than 100 per cent certain that a CS will be followed by a given US. The degree of this conviction, which might be likened to wagering, is independent of the intensity of either event. In short, we interpret $V$ to be an index of belief, prediction, or inferred causality, and that, as such, it represents the reliability of an internodal link within an associative network. Moore and Stickney (1985, p. 228) discuss some of the consequences and limitations of this conceptualization regarding the asymptote of learning. The issue is largely irrelevant for present purposes except insofar as it bears on the number of degrees of freedom available for describing phenomena.

Whereas $\theta$ is treated as a constant within a given application of Equation 1, $\alpha$ can vary from trial to trial. This variation in $\alpha$ accounts for most, if not all, of the phenomena that prompted development of the R–W model, e.g., blocking and overshadowing. The parameter $\alpha$ also appears in the R–W model, but it is typically treated as a constant because the mechanism that predicts these phenomena resides in the zero-sum rule (except see Wagner, 1978).

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The P-H model expresses the increments in associative value accompanying acquisition by the expression

$$\Delta V = S \alpha \lambda \qquad (2)$$

As in the M–S model, $\alpha$ varies according to rules that are sufficient in themselves to predict blocking and overshadowing, as well as simple acquisition and other phenomena.

Although both models share the R–W model's ability to predict many of the better established and more highly visible phenomena of classical conditioning, the rules for computing $\alpha$ in the two models differ profoundly. Mackintosh (1975) and M–S assume that $\alpha$ for a CS *increases* from one reinforced trial to the next, provided it is the best predictor of the US (largest $V$) among all stimuli, including CSs and the context, occurring at the same time. On these occasions, $\alpha$s for stimuli with smaller $V$s decrease, and therefore their capacities to further strengthen associative links to the US are diminished. Thus, changes in $\alpha$s for a set of CSs depend on their relative $V$s with respect to the US. By contrast, the P–H model assumes that $\alpha$ for a CS that is consistently paired with a US *decreases* over trials. Despite this difference in assumptions regarding variations in $\alpha$, the two models give qualitatively similar predictions in many protocols.

The two models are similar in another respect: Both the M–S and P–H models assume inhibitory learning *in parallel* with acquisition of $V$. It is therefore possible for a given CS to possess both excitatory and inhibitory associations concurrently, the two associations summating algebraically to determine CR strength. By contrast, the R–W model assumes that a CS can possess either an excitatory or an inhibitory association with the US, but not both simultaneously. Moore and Stickney (1982, 1985) refer to "negative" learning as the acquisition of an antiassociation; Pearce and Hall (1980) use the term inhibitory conditioning. We use the symbol $N$ to denote the inhibitory variable in the two models.

## Shortcomings in Brief

Although the M–S model successfully describes many facets of conditioned and latent inhibition, it fails to predict realistic scenarios under partial reinforcement, extinction, and related protocols involving nonreinforced trials. Our proposed solution involves changing the expressions for computing decreases of $V$ and increases of $N$ that are applied on nonreinforced trials. The P–H model has problems describing partial reinforcement, and these have been discussed by its originators (Pearce, Kaye & Hall, 1982). The model predicts nonmonotonic relationships between the percentage of trials that are reinforced and the level of conditioning achieved. Another shortcoming of the P–H model is that terminal values of associative strength are highly dependent on the length of sequences of reinforced and nonreinforced trials. One proposed solution involves alternative rules for computing $\alpha$. Another solution involves changes in rules for computing $V$ and $N$.

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# Moore and Stickney Model

Moore and Stickney (1980) developed their model originally in order to place Mackintosh's (1975) theory on a firm computational footing. Summarizing the model as described in a recent chapter (Moore & Stickney, 1985): (a) *Associative value*, $V$, represents the prediction of the US by a CS. (b) Antiassociative value, $N$, represents the prediction of nonreinforcement by a CS. (c) The strength of a CR to a given CS depends on its net associative value, $\dot{V}$, given by $V - N$. (d) $V$ for a given CS increases when it accurately predicts the US and decreases otherwise. (e) By contrast, $N$ increases when the sum of $V$s for all CSs present is above some threshold, and the US does not occur. $N$ decreases whenever the US does occur. (f) Changes of $V$ and $N$ depend on the associability, $\alpha$, of the CS. (g) $\alpha$ increases to the extent that the CS is the best predictor of the US than other stimuli (including itself) in the situation. Otherwise, it decreases. (h) This dependence of $\alpha$ on the predictive associative relationships among all stimuli in the situation implies the existence of a network of $V$s and $N$s. (i) The model applies to real time, implying that computations occur continuously, both within and between trials.

In the following formal statement of the M–S model, subscripts are used to specify stimuli in the role of predictor. Superscripts denote the target of the prediction. Thus, if $A$ and $B$ are two stimuli such as a CS and US, then $V_A^B$ designates the associative value of $A$ *predicts* $B$. $N_A^B$ designates the antiassociation $A$ *predicts not* $B$, and $(V_A^B - N_A^B) = \dot{V}_B^A$ is the net value of the relationship. It is important to note that $V_A^B$ does not equal $V_B^A$.

Generalizing the notation, when the $i$th CS is accompanied or followed by a $k$th event, the associative value between $CS_i$ and event $k$, $V_i^k$, is increased by

$$\Delta V_i^k = \alpha_i\, \theta\, \tau\, (1 - V_i^k) \tag{3}$$

When event $k$ does not occur, the associative value between $CS_i$ and the event $k$, $V_i^k$, is decreased by

$$\Delta V_i^k = \alpha_i\, \theta'\, \tau\, (0 - V_i^k) \tag{4}$$

The parameter $\alpha_i$ is the associability of the $i$th CS; it ranges between 0 and 1. The parameter $\theta$ $(0 < \theta \le 1)$ is the rate of change in the association when the reinforcer is presented, and $\theta'$ $(0 < \theta' < \theta)$ is the rate of change of the association when the reinforcer is not presented.

The parameter $\tau$ is a function of time such that

$$\tau = e^{k(q - \Delta t)} \tag{5}$$

where $q$ is a constant equal to the optimal interval for association, $\Delta t > 0$ is the interval between the $i$th CS and the $k$th event in $q$ steps, and $k$ is a constant $(0 < k < 1)$. In our

simulations, $\tau$ with $\Delta t < q$ was set equal to .067. (Refer to Schmajuk & Moore, 1985 for a fuller explanation of implementation).

The rule for increasing the antiassociation $N_i^k$ between $CS_i$ and event $k$ is as follows: whenever $CS_i$ is neither accompanied nor followed by the event $k$ *and* the sum of the associative values of all CSs present, $\sum \dot{V}_j^k$, exceeds an arbitrary and constant threshold $L$, antiassociative value, $N_i^k$, is increased by

$$\Delta N_i^k = \alpha_i \theta' \tau (1 - N_i^k) \tag{6}$$

whenever $\sum_j V_j^k > L$. The rule for decreasing $N_i^k$ is as follows: whenever the $k$th event follows the $CS_i$ the antiassociative value decreases by:

$$\Delta N_i^k = \alpha_i \theta \tau (0 - N_i^k) \tag{7}$$

The net associative value of $CS_i$ with respect to event $k$ is $\dot{V}_i^k = V_i^k - N_i^k$.

In the M–S model, associability of a CS depends on associative processes. The associability of $CS_i$, $\alpha_i$, may increase, decrease, or remain unchanged according to a weighted combination of event-specific components, $\Delta \alpha_i^k$. These event-specific components are computed based on the relationship between the associative value of $CS_i$ and the event $k$ and the associative value of another CS, $CS_j$, with the same event $k$. Whenever $CS_i$ and $CS_j$ are present together with the $k$th event and provided $V_i^k > V_j^k$,

$$\Delta \alpha_i^k = c(1 - \alpha_i^k)(V_i^k - V_j^k) \tag{8}$$

$V_i^k$ always corresponds to the second highest associative value with respect to event $k$ of all stimuli present with $CS_i$, including the context and/or the US.

If $V_i^k \le V_j^k$,

$$\Delta \alpha_i^k = c(0 - \alpha_i^k)(V_j^k - V_i^k) \tag{9}$$

where $V_j^k$ is the highest associative value with respect to $k$ of all stimuli present with $CS_i$. The parameter $c$ in Equations 8 and 9 is set $0 < c < 1$. Once the event-specific components of $\alpha_i$ have been computed, they are combined in the expression

$$\Delta \alpha_i = \sum_j \phi_j \Delta \alpha_i^j / \sum_k \phi_k \tag{10}$$

The weight assigned to each $\Delta \alpha_i^j$ is indicated by the constant $\phi_j$. The sum over the index $j$ in the numerator of Equation 10 involves all the events present on that trial or time step. The sum over the index $k$ in the denominator is over all the events or stimuli the subject has encountered in the context, even though some of these may not be present at the time that $\Delta \alpha_i$ is computed. Thus, the numerator of Equation 10 involves associations among

stimuli that are present at the moment of computation, whereas the denominator involves the weights of these stimuli plus those encountered previously. The US is presumed to be represented in memory more strongly than are CSs, which are in turn typically weighted more heavily than the context.

## Partial Reinforcement and Extinction

Shortcomings of the M-S model came to light in simulations of certain protocols involving nonreinforcement: partial reinforcement, extinction, and simple differential conditioning. Although it is unreasonable to require that any model be universally applicable to all tasks and circumstances, the current versions of both models are so widely at variance with the experimental literature that corrective measures seemed called for. This is particularly the case regarding partial reinforcement during acquisition. The experimental literature suggests that partial reinforcement in classical conditioning results either in a lower level of CR strength (e.g., CR frequency) than that obtained under 100% reinforcement or else a level that is just as high as in the 100% case. Once acquired, a CR can be maintained at close to full strength with schedules of reinforcement as lean as 5% (Gormezano, Kehoe, & Marshall, 1983). Gormezano and Moore (1969) tabulated 7 of 15 studies across a range of species and preparations in which 50% partial reinforcement resulted in levels of CR strength following acquisition that were significantly below those observed under 100% reinforcement. No difference was noted in the remaining 8 studies. This much seems clear: Levels of conditioned responding under partial reinforcement ought neither to exceed those under 100% reinforcement, appetitive instrumental conditioning tasks being a well known exception (Kimble, 1961), nor be so low as to portend imminent extinction.

## Simulations with the M-S Model

The following simulation experiments all assumed the $V$ and $N$ for both the CS and the context have initial values of 0 prior to any training. The initial value of $\alpha$ for the CS was .5; that of the context was .1. The parameter $c$ in Equations 9 and 10 was .3, and $\theta$ and $\theta'$ in Equations 3 and 4 were .1 and .01, respectively. Following Moore and Stickney (1980), the weights $\phi_j$ and $\phi_k$ in Equation 10 were 1.0 for the US, .16 for CSs, and .01 for the context. Figure 1 illustrates the M-S applied to 50% partial reinforcement. It shows that acquisition of $V$ as a function of trials with a 50% reinforcement protocol initially increases and then decreases to a stable level well below that predicted with the same parameter set for 100% reinforcement. A portrayal more in keeping with the literature would show $V$ under 50% reinforcement increasing uniformly and leveling off to a point just below that obtained with 100% reinforcement. As Fig. 1 makes clear, the problem arises from unrestrained development of $N$ once the threshold for triggering Equation 6

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of $\Lambda$ ... Although extended extinction training ... ... ...
decreases of associability, most researchers now recognize that ... ...
not necessarily install inhibitory properties in the same sense as ... ...
training and that an extinguished CS can retain considerable ... ... ...
e.g. Hendry, 1982.

Simple differential conditioning with one CS consistently reinforced ($CS+$) ... ...
and uncompounded CS consistently not reinforced ($CS-$) presents ... an other difficulty
for the M S model. Assuming there is only negligible stimulus generalization from $CS+$
to $CS-$, $CS-$ can generate $N$ only when $V$ for the context is above threshold. Thus recall-
ing that $V$ for context continuously undergoes extinction by Equation 4 during intertrial
intervals, $CS-$ in simple differential conditioning can only generate $N$ (and negative $V$)
when the threshold $L$ is arbitrarily low. Although this resistance to generating $N$ might be
desireable in some applications, the parameter $L$ can be dispensed by allowing $N$ to vary
continuously with $V$. This can be accomplished by substituting the following expression
for Equation 6

$$\Delta N_i^k = \alpha_i \theta' \tau (1 - N_i^k)(\sum V_j^k - \sum N_j^k) \tag{11}$$

The factor $\sum V_j^k - \sum N_j^k = \sum V_j^k$ represents the difference between $V$'s and $N$'s of all the
CSs present on the trial, including $CS_i$. The addition of the same factor to the expression
for decreasing $V$, in addition to restraining undesireable reduction of $V$, also introduces
symmetry to the rules for changing $V$ and $N$. We therefore replace Equation 4 by the
following expression:

$$\Delta V_i^k = \alpha_i \theta' \tau (0 - V_i^k)(\sum V_j^k - \sum N_j^k) \tag{12}$$

Figures 2-4 show that the new expression for decreasing $V$ and increasing $N$ allow the
M S model to more accurately describe partial reinforcement (Fig. 2), extinction (Fig.
3) and reacquisition (Fig. 4). In the case of partial reinforcement, Fig. 2 shows that the
revised model allows normal appearing monotonic acquisition of $V$ to a level below that
obtained under continous reinforcement.

Figure 3 shows that $V$ in extinction can go below zero, but not so dramatically as
before. The change also implies that reacquisition would normally be more rapid than
original acquisition (see Fig. 4), in agreement with some of the literature (Scavio, Ross,
& McLeod, 1983). In the case of the rabbit nictitating membrane (NM) response, rapid

reacquisition may not be specific to the target CS because of a general transfer process in which initial acquisition to one CS promotes rapid acquisition to another, quite distinct CS (Kehoe, Morrow, & Holt, 1984).

It should be noted that neither the older or here–modified version of the M–S model generates spontaneous recovery. This criticism applies to all contemporary models, including P–H and R–W. Exploring the full implications of this modification of the M–S model lies beyond the scope of this article. However, simulation experiments indicate that the descriptive power of the model remains largely intact when Equations 11 and 12 are employed instead of Equations 4 and 6 in protocols involving nonreinforced trials, most notably conditioned and latent inhibition.

## Pearce and Hall Model

Pearce and Hall (1980) proposed their model as an alternative to Mackintosh-type attention theories because of their discovery that a series of acquisition trials with a weak shock US in a conditioned suppression task can retard subsequent acquisition using a stronger shock US (Hall & Pearce, 1979), a phenomenon they liken to latent inhibition (LI). Negative transfer (NT) due to initial training with a weak US falls naturally out of the P–H model by virtue of the assumption that a CS's associability decreases with repeated pairings with a US. Although NT from a weak to a strong US in conditioned suppression has been replicated by others, it is not always obtained in such studies; nor is there evidence for the effect in the rabbit NM response preparation where positive transfer seems the rule (Ayres, Moore, & Vigorito, 1984). Such positive transfer does not disprove the model because it is predicted whenever the initial value of $\alpha$ is relatively small and the US in the first phase of training produces a relatively high level of $V$. Negative transfer is the surprising result; it is predicted whenever the first-phase US yields only a low level of $V$ and the initial value of $\alpha$ is large (see Ayres et al, 1984, for elaboration of this point). Initial values of $\alpha$ presumably depend on generalization from other similar stimuli outside the training context (Pearce & Hall, 1980, page 538).

In addition to predicting NT, the P–H model provides a mechanism for conditioned inhibition, thereby filling a void in previous attention theories. It was only after their model first appeared that Moore and Stickney (1982; 1985) incorporated conditioned inhibition into their Mackintosh-type model.

We now summarize the P–H model as we understand it: (a) The excitatory component of the associative relationship between a CS and US, $V$, increases whenever the CS's associability, $\alpha$, is greater than zero and the intensity of the US on a given trial, $\lambda$, is larger than that predicted by all the CSs present on that trial; $V$ never decreases. (b) The inhibitory associative component, $N$, increases whenever $\alpha$ is greater than zero and the intensity of the US on a given trial is less than or equal to that predicted by all the CSs

present on that trial. Like $V$, $N$ for a given CS can never decrease. (c) Net associative strength equals the sum of the differences between the $V$s and $N$s of all the CSs present on the trial, $(V - N)$. We denote this net associative value as $\bar{V}$, in keeping with the notation used in the M-S model; it is the net prediction of a US equal to $\lambda$ by all stimuli present on a given trial. (d) $\alpha$ for computations of $V$ and $N$ for all CSs present on a given trial equals the absolute value of the difference between $\lambda$ of the preceding trial and $\sum \bar{V_j}$ of the current trial. (e) Letting $\alpha$ equal the geometric mean of the $\alpha$s for previous trials is a permissible option under the model (see, e.g., Kaye & Pearce, 1984).

Stated formally, whenever the intensity of the US presented on a given trial is larger than the sum of the $V$s of all CSs present on that trial, $V$ increases according to

$$\Delta V_i = S_i \, \alpha_i \, \lambda \tag{13}$$

$S_i$ is the salience of the $CS_i$, $\alpha_i$ is its associability, and $\lambda$ is the intensity of the US ($S_i$, $\alpha_i$, and $\lambda > 0$). Whenever the intensity of the US presented on a given trial is less than the sum of $V$s of all CSs present on that trial, $N$ increases according to

$$\Delta N_i = S_i \, \alpha_i \dot{\lambda} \tag{14}$$

$\dot{\lambda} = \sum \dot{V_j} - \lambda$ where $\sum \dot{V_j}$ is the sum of net associative values of all CSs, including $CS_i$ acting on Trial $n - 1$. Equations 13 and 14 imply that when the intensity of the reinforcer is less than $\dot{V_i}$, $V_i$ does not decrease, but rather $N_i$ increases until it reaches the same value as $V_i$. When the sum of $V$s of all CSs equals $\lambda$, Equation 14 rather than Equation 13 should be applied in order that $V_i$ remains unchanged.

The value of $\alpha_i$ on Trial $n$ for all CSs acting on Trial $n - 1$, is given by

$$\alpha_i^n = | \lambda^{n-1} - \sum \dot{V_j}^{n-1} | \tag{15}$$

The expression $\sum \dot{V_j}^{n-1}$ represents the prediction of the US by all CSs, including $CS_i$ present on Trial $n - 1$. When the reinforcer is accurately predicted by all the CSs present on Trial $n - 1$, $\alpha_i$ becomes zero on Trial $n$. Equation 15 cannot be used to determine $\alpha_i$ on the first trial in which $CS_i$ is presented, and some initial value must be assigned. When $CS_i$ is presented on a second occasion, however, $\alpha_i$ is determined by Equation 15.

## Simulations with the P–H Model: Partial Reinforcement

Pearce et al (1982) point out that the original model has difficulty describing acquisition under partial reinforcement. For example, no conditioning is predicted when reinforced and nonreinforced trials are alternated if the sequence begins with a nonreinforced trial. In this case, on Trial 1 both $\lambda$ and $V_i$ equal zero, and therefore $\alpha_i$ on Trial 2 is zero. On

Trial 2, when the US is presented, $\lambda = 1$, but since $\alpha_i = 0$, no increase in $V$ can occur. In order to solve this problem, Pearce et al (1982) suggested using the geometric mean of values of $\alpha_i$ computed on previous trials:

$$\alpha_i^n = \gamma \mid \lambda^{n-1} - \sum V_j^{n-1} \mid + (1 - \gamma) \, \alpha_i^{n-1} \tag{16}$$

The parameter $\gamma$ is between 0 and 1. Equation 16 yields Equation 15 when $\gamma = 1$.

Problems for the model under partial reinforcement are not entirely corrected by Equation 16, as illustrated in Fig. 5. Figure 5 shows simulated net associative values $(\dot{V})$ for a single CS as a function 50% and 80% randomly reinforced trials. Notice that the asymptotic value of $\dot{V}$ with 50% is higher than that predicted with 80% reinforcement, and that the asymptotic value of $\dot{V}$ with 80% reinforcement is higher than that obtained with 100% reinforcement. (Starting values of $V_i$ and $N_i$ were 0; $\alpha_i = 1$ initially, $\gamma = .5$ or 1, $\lambda = 1$, and the initial value of $S_i = 1$.) In attempting to rearrange these asymptotic levels so that higher levels of conditioning correspond to higher percentages of reinforcement, Pearce et al (1982) introduced additional rate parameters into Equations 13 and 14. These parameters are denoted $\beta_E$ and $\beta_I$ for changes in excitatory and inhibitory association, respectively, and they are bounded between 0 and 1. Equation 13 becomes

$$\Delta V_i = S_i \, \alpha_i \, \beta_E \, \lambda \tag{17}$$

and Equation 14 becomes

$$\Delta N_i = S_i \, \alpha_i \, \beta_I \, \bar{\lambda} \tag{18}$$

Using computer simulations, Pearce et al. (1982) showed that when $\beta_E < \beta_I$ the growth of $\dot{V}$ with 50% reinforcement reaches an asymptote lower than that attained with 100% reinforcement. Equations 17 and 18 yield a lower asymptote for $\dot{V}$ because on reinforced trials $V$ increases less than $N$ does on nonreinforced trials. Our simulations confirm this point. However, Fig. 5 suggests that when growth of $\dot{V}$ with 80% reinforcement is adjusted to levels close to those predicted for 100% reinforcement, by adjusting $\beta_E$ and $\beta I$, the asymptotic value of $\dot{V}$ attained with 50% reinforcement is too low. This is so because with 80% reinforcement $\beta_E$ needs to be much smaller than $\beta_I$, and this combination of $\beta$s does not allow $V$ to grow enough with 50% reinforcement. Therefore, the introduction of additional rate parameters, as proposed by Pearce et al. (1982) does not yield appropriate asymptotes for $\dot{V}$ with different percentages of reinforcement.

## Alternative Forms of the Pearce and Hall Model

In order to improve the P–H model's rendering of partial reinforcement, we considered two alternative forms of the model. The first computes $\alpha$ on the basis of the outcome of

Trial $n$ instead of the outcome of Trial $n - 1$ as in Equation 15. That is, $\lambda^{n-1}$ in Equation 15 is replaced by $\lambda^n$:

$$\alpha_i^n = |\ \lambda^n - \sum V_j^{n-1}\ |$$ (19)

Equations 13 and 14 remain unchanged. Stated somewhat anthropomorphically, Equation 19 implies that the subject waits for the outcome of a trial before deciding by what amount to increment $V$ by Equation 13 or $N$ by Equation 14. The general form of $\alpha_i$ in the revised model is given by the expression

$$\alpha_i^n = \gamma\ |\ \lambda^n - \sum \dot{V}_j^{n-1}\ | + (1 - \gamma)\alpha_i^{n-1}$$ (20)

Unrestricted use of Equation 20 does not correct the prediction of higher asymptotic levels of responding under partial reinforcement than under 100% reinforcement. The desired result necessitates that $\alpha$ be computed with either Equation 20 ($\gamma < 1$) or Equation 19 ($\gamma = 1$), *whichever yields the smaller value of $\alpha$* . Without this restriction the model predicts higher $V$ with 80% reinforcement than with 100%, as did the original P–H model. Figure 6 shows the simulated $V$ for a single CS as a function of 50% and 80% reinforced trials, using Equation 20 with $\gamma = 1$ and .5. In the latter case ($\gamma = .5$) the above mentioned restriction was applied. With either $\gamma$, this restricted-$\alpha$-version of the P–H model yields asymptotic levels of responding for 50% and 80% reinforcement that are (a) lower than that predicted for 100% reinforcement, (b) sufficiently high, and (c) in the appropriate order.

In the second alternative form of the model, Equations 13 and 14 are replaced by a single equation expressing the changes in $\dot{V}$ instead of separate changes of $V$ and $N$.

$$\Delta \dot{V}_i^n = S_i \alpha_i (\lambda^n - \sum \dot{V}_j^{n-1})$$ (21)

Equation 21 implies that $\dot{V}$ converges to $\lambda$, increasing when $\lambda$ increases, and decreasing when $\lambda$ decreases. Equation 21 may be regarded as the R–W model with the addition of a modifiable associability term. It is similar to the expression proposed by Wagner (1978) to encompass CS preexposure effects within the framework of the R–W model. The expressions for changes in $\alpha$ are the same as in the original model, i.e., Equations 15 or 16 apply.

Figure 7 shows the simulated $\dot{V}$ of a single CS as a function of 50% and 80% reinforced trials using Equation 21 and with both rules for computing $\alpha$. As in the case of the first alternative version of the model, Equation 21 predicts asymptotic levels for 50% and 80% reinforcement that are (a) lower than that predicted for 100% reinforcement, (b) sufficiently high, and (c) in the appropriate order. However, Equation 16 yields a higher level of responding than Equation 20 with 80% reinforcement.

Figure 8 summarizes the predictions made by the original and two alternative versions of the model for a wide range of percentages of reinforcement. Rates of reinforcement in

the 10% to 50% range were obtained by introducing the required number nonreinforced trials between two reinforced trials. Rates of reinforcement in the 66% to 90% range were obtained by introducing the required number of reinforced trials between two nonreinforced trials. When the original P-H model (Equation 16) is applied with $\beta_E = \beta_I = .1$, any rate of reinforcement from 50% and over exceeds the asymptotic level obtained with 100% reinforcement. When $\beta_E = .015$ and $\beta_I = .10$, only rates of reinforcement of 90% or more achieve sufficiently high asymptotes; asymptotes with lower reinforcement probability are too low to agree with empirical expectations. The two alternative versions of the model yield asymptotic levels of net associative strength that are more realistic. In these cases the relationship between asymptotic associative strength and reinforcement probability is in closer agreement with empirical expectations, tending to lie on a line with slope equal to 1.

Experiments in pigeon autoshaping have shown that partial reinforcement can produce higher levels of responding than continuous reinforcement (Gibbon, Farrell, Locurto, Duncan, & Terrace, 1980). Gibbon et al.'s results show that response rate monotonically decreases with increasing probabilities of reinforcement. Neither the original nor the revised versions of the P-H model can account for this phenomenon. Simulations with the original P-H model with $\beta_E = \beta_I = .1$ show that $\dot{V}$ first increases and then remains constant with increasing rates of reinforcement (Fig. 8). Simulations with the revised models show that $\dot{V}$ increases with increasing rates of reinforcement (Fig. 8). According to Gibbon et al. (1980) the effect of partial reinforcement on response rate parallels the well-established effect of partial reinforcement on instrumental learning, and might be explained in terms of the frustration generated by nonreinforced trials (Amsel, 1962).

In addition to the problem of inappropriate asymptotic levels of net associative strength under partial reinforcement, the original P-H model is severely path dependent. That is, terminal levels of $\dot{V}$ depend on the sequential pattern of reinforced and nonreinforced trials. Path dependency is a concern only in tasks in which the asymptotic level of conditioned responding is known to be sensitive to the percentage of trials that are reinforced but relatively insensitive to the sequential structure that underlies that percentage. In the case of classical aversive conditioning, such as the eye blink in humans and rabbits, for example, asymptotic performance levels are not particularly sensitive to the sequential properties of trials (except see, Hoehler & Leonard, 1973).

Path dependency of the original and alternative versions of the P-H model is contrasted in Table 1. The entries are the average $\dot{V}$ on the last 10 trials following 300 trials of patterned 50% reinforcement in which runs of reinforced trials were alternated with equally long runs of nonreinforced trials. Initial parameterization was the same as in Fig. 8. Equation 16 of the original model yields the greater path dependence, as indexed by the range of entries under the first column (.11). The revised forms of the model (Equations 13 and 14, with $\alpha$ computed with Equation 20, and Equation 21, with $\alpha$ computed with

either Equation 16 or Equation 20) reduce this range to .03 and .02, respectively. Thus, path dependence under partial reinforcement is substantially reduced by using either of the alternative forms of the model instead of the original.

## Implications for Latent Inhibition and Negative Transfer

As indicated above, the original P-H model (Equation 16) predicts both LI and NT. With $\gamma = 1$ in Equation 16, LI occurs with a single CS preexposure. Retarded acquisition is predicted because CS presentation in the absence of the US produce zero associability, thereby preventing any increase in $V$ on the first reinforced trial. By contrast, NT requires that a sufficient number of CS–US pairings have occurred to decrease $\alpha$ to zero. Conditions leading to NT are (a) low $\lambda$ in the first phase of training, (b) high initial $\alpha$, and (c) a low value of $\gamma$. Positive transfer is likely if any one of these conditions is not satisfied. When $\gamma = .5$ in Equation 16, both LI and NT require more than a single CS presentation prior to acquisition with a strong US to reflect the effect of Stage 1 trials. In both instances retarded acquisition with a strong US in the second phase of training comes about because CS presentations in the first phase cause $\alpha$ to decrease to zero, thereby producing a comparatively small average $\alpha$ during early reinforced trials.

Table 2 shows $\dot{V}$ on the first trial following Stage-1 latent inhibition (LI) and negative transfer (NT) paradigms as predicted by the original and alternative forms of the model. The original version of the model (P-H $(n - 1)$), with $\alpha$ defined according to Equation 16, yields LI and NT with $\gamma = .5$ or 1. Table 2 shows that the first alternative version of the model (P-H $(n)$), with $\alpha$ defined according to Equation 20, yields LI and NT only when $\gamma = .5$. As in the original model, retarded acquisition in the second phase of LI and NT results from reduced $\alpha$ on early reinforced trials. Both LI and NT reflect the number of CS presentations on the first stage of training. The behavior of the second alternative form of the model (P-H$_{R-W}(n - 1))XS$, with $V$ computed by Equation 21, depends on whether $\alpha$ is computed with Equation 16 or Equation 20. With Equation 16, LI and NT are predicted with $\gamma = .5$ or 1. With Equation 20, LI and NT are predicted only when $\gamma = .5$.

## One-Trial Blocking

Unlike the R–W model, the models considered here do not allow for blocking on the first compound-CS trial following Stage-1 training to a single CS. The question of whether blocking occurs on the first Stage-2 trial has been the focal point of experimental efforts determine which type of theory is to be preferred. Until recently, most available evidence suggested that blocking requires at least two Stage-2 trials to occur (e.g., Mackintosh, Dickinson, & Cotton, 1980). More recent evidence on the question suggests that one-trial

blocking, as anticipated by the R-W model, can occur under some circumstances (Balaz, Kasprow, & Miller, 1982; Dickinson, Nicholas, & Mackintosh, 1983).

The original P-H model does not permit one-trial blocking, and in this respect it resembles the M-S model. Using Equation 16 to compute $\alpha$, one-trial blocking is not possible because at least one compound-CS trial is necessary in order to reduce the added CS's initial value of $\alpha$ to the near-zero value implied by the presence of the previously conditioned CS. However, if $\alpha$ is computed by Equation 20, one-trial blocking is possible because the one-trial delay does not arise. Equation 21 allows for one-trial blocking, independent of the expression used to compute $\alpha$, for the same reason that the the R-W model predicts one-trial blocking, i.e., because the expression $(\lambda - \sum V_j)$ can be near 0 on the first compound trial provided $\sum V_j$ of the Stage-1 CS is near $\lambda$.

## General Discussion

The M-S and P-H models fail to provide acceptable renderings of acquisition under partial reinforcement and certain other phenomena. Modifications of these models alleviate these shortcomings while retaining their basic assumptions and predictive power (see Schmajuk & Moore, 1985). The revised version of the M-S model presented here gives improved predictions for extinction, reacquisition, partial reinforcement, and simple two-CS differential conditioning. Regarding the P-H model, the two approaches to improved performance were considered. Both provide reasonably good renderings of acquisition under partial reinforcement, with appropriate asymptotic levels of net associative value and suppression of path dependency. In addition, both alternative forms of the P-H model are able to predict one-trial blocking. Because it involves only a minor change in the computation of a CS's associability, the first alternative form of the P-H model more closely resembles the original than does the second alternative. The second version changes the computation of excitatory associative value so as to place the P-H model into the same family as the R-W model and the Sutton-Barto model (Sutton & Barto, 1981). The second version is interesting because the discrepancy between the actual outcome of a trial and the anticipated outcome, obtained by summing the predictions of all CSs present on that trial, determines both $\alpha$ and asymptotic values of net associative strength. This tactic is similar to one proposed by Frey and Sears (1978) in which the attentional variable represents the information value of a CS in terms of its recent associative value, and it allows the model to predict latent inhibition. In order to predict latent inhibition without recourse to an attentional variable, Wagner (1978) proposed that changes in the CS effectiveness might be represented in the R-W model by the inclusion of a variable reflecting how well the CS is predicted by stimuli that precede it.

Our suggested revisions of the two models might be challenged as being entirely arbitrary. This is not the case, as they were arrived at largely through a process of trial

and error in which various remedial approaches were implemented into a wide range of simulation protocols that included simple acquisition, blocking, conditioned inhibition, differential conditioning, extinction, latent inhibition, and overshadowing. This process of trial and error emphasized a point about mathematical models that is often overlooked by their detractors: Mathematical structure, not simply number of parameters or degrees of freedom, dictate the descriptive power of a model.

We do not deny the possibility that other modifications will be discovered that prove preferable; we have simply not discovered any that retain the basic mathematical character of the originals without introducing new constructs. Revisions more drastic than those considered here might take the form of attentional or hybrid models that possess the best features of the M-S, P-H, and R-W models. One clue that this might be possible is suggested in the revised M-S model, in which the threshold for triggering antiassociations is replaced by a mechanism very similar to that used by the P-H model to generate conditioned inhibition. Another clue is suggested in the second revised version of the P-H model, (Equation 21) which is essentially a R-W model but with a mechanism for controlling CS associability. In this respect it can be classed with the Frey and Sears (1978) model.

Simulation experiments with alternative versions of the M-S and P-H models should ultimately point the way to further refinements and better specification of the appropriate domains for each. We are a long way from declaring a clear preference for either model, and the experimental literature suggests that each may have its place. As a class, P-H models may be most appropriate for characterizing events in the domain of conditioned suppression and perhaps, more generally, in systems involving autonomic-like processes of arousal and orienting (Kaye & Pearce, 1984). The revised M-S model may be more appropriate in the domain of discrete skeletal responses such as the rabbit NM response (Ayres et al, 1984). Wherever further explorations of these models may lead, we believe our approach illustrates some of the benefits to theory construction and assessment to be derived through simulation experiments over a broad range of training scenarios. Comparisons among competing theories can be sharpened without recourse to experimentation. Although real experiments always have a place in choosing among competing theories, simulation experiments can guide decisions regarding protocols that are most likely to resolve such choices.

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**TABLE 1. Pearce–Hall Rules for Computing Associability: Average Net Associative Value on the last 10 Trials following 300 Trials Consisting of Alternating Runs of Reinforced and Nonreinforced Trials**

| Sequences | Algorithm | | | |
|---|---|---|---|---|
| | P–H $(n-1)$ | P–H$(n)$ | P–H$_{R-W}(n-1)$ | P–H$_{R-W}(n)$ |
| 10 | .08 | .66 | .50 | .50 |
| 1100 | .10 | .65 | .49 | .49 |
| 111000 | .12 | .65 | .51 | .51 |
| 11110000 | .17 | .65 | .51 | .51 |
| 1111100000 | .19 | .63 | .50 | .50 |
| Range | .11 | .03 | .02 | .02 |

**Note.** The basic sequence, repeated over the 300 trials, is indicated as series of 1 (reinforced) and 0 (nonreinforced) trials. P–H $(n-1)$ refers to the original rule for computing $\alpha$ with equation 16. P–H $(n)$ refers to the alternative rule for computing $\alpha$ with Equation 20, with $\gamma = 1$ when $\alpha_i^{n-1} > \alpha_i^n$. P–H$_{R-W}(n-1)$ refers to the alternative rule for computing $\dot{V}$ with Equation 21 and $\alpha$ with Equation 16. P–H$_{R-W}(n)$ refers to the alternative rule for computing $\dot{V}$ with Equation 21 and $\alpha$ with Equation 20.

**TABLE 2. Pearce–Hall Rules for Computing Associability: Net Associative Value After One Reinforced Trial Following 5 or 10 Trials of Either CS-alone or CS paired with a weak US in Stage 1.**

| | Algorithm | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| | P–H $(n-1)$ | | P–H$(n)$ | | P–H$_{R-W}(n-1)$ | | P–H$_{R-W}(n)$ | |
| Gamma | 1 | .5 | 1 | .5 | 1 | .5 | 1 | .5 |
| Control | .50 | .50 | .50 | .50 | .50 | .50 | .50 | .50 |
| LI (5) | .00 | .02 | .50 | .26 | .00 | .03 | .50 | .26 |
| LI (10) | .00 | .02 | .50 | .25 | .00 | .00 | .50 | .25 |
| NT (5) | .11 | .12 | .51 | .29 | .09 | .10 | .50 | .30 |
| NT (10) | .10 | .10 | .52 | .30 | .08 | .09 | .50 | .29 |

**Note.** The number of trials on the first phase of LI or NT is indicated in parenthesis. Control groups received neither CS preexposure nor CS paired with a weak US. In NT, $\lambda = .1$ during the first phase of training. P–H $(n-1)$ refers to the original rule for computing $\alpha$ with Equation 16. P-H $(n)$ refers to the alternative rule for computing $\alpha$ with Equation 20, with $\gamma = 1$ when $\alpha_i^{n-1} > \alpha_i^n$. P–H$_{R-W}$ $(n-1)$ refers to the alternative rule for computing $V$ with Equation 21 and $\alpha$ with Equation 16. P–H$_{R-W}(n)$ refers to the alternative rule for computing $V$ with Equation 21 and $\alpha$ with Equation 20.

## Figure Captions

**Figure 1.** Partial reinforcement with the Moore–Stickney model: $V$, $N$, and Net associative value $\dot{V}$ , as a function of trials for 100% and 50% random reinforcement. $V$ and $N$ with 50% reinforcement are plotted separately.

**Figure 2.** Partial reinforcement with the modified Moore–Stickney model: $V$, $N$, and Net associative value $\dot{V}$ , as a function of trials for 100% and 50% random reinforcement. $V$ and $N$ with 50% reinforcement are plotted separately.

**Figure 3.** Extinction with the original and revised Moore–Stickney model: Net associative value $\dot{V}$ as a function of trials.

**Figure 4.** Reacquisition with the original and revised Moore–Stickney model. Initial acquisition (100% reinforcement) with the original Moore– Stickney model is shown in FIgs. 1-2.
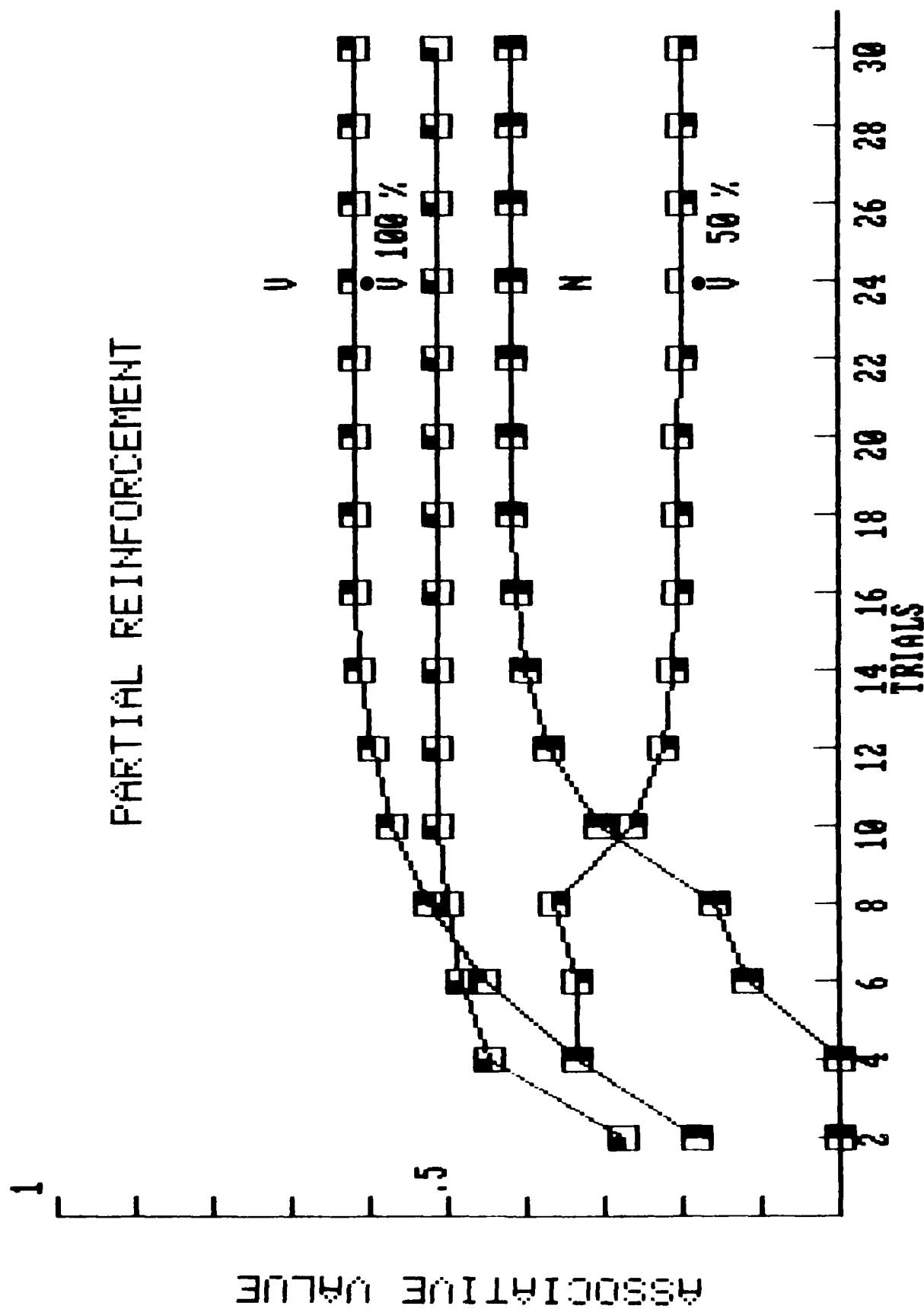
**Figure 5.** Partial reinforcement with the original Pearce–Hall model. Net associative value as a function of trials under 50% (alternated reinforced and nonreinforced trials) and 80% (four reinforced followed by one nonreinforced trial) reinforcement. (1): $\beta_E = \beta_I = .5$; (2) $\beta_E < \beta_I.\beta_E = .1, \beta_I = .5$. Dashed line indicates asymptote reached with a continuous reinforced schedule.

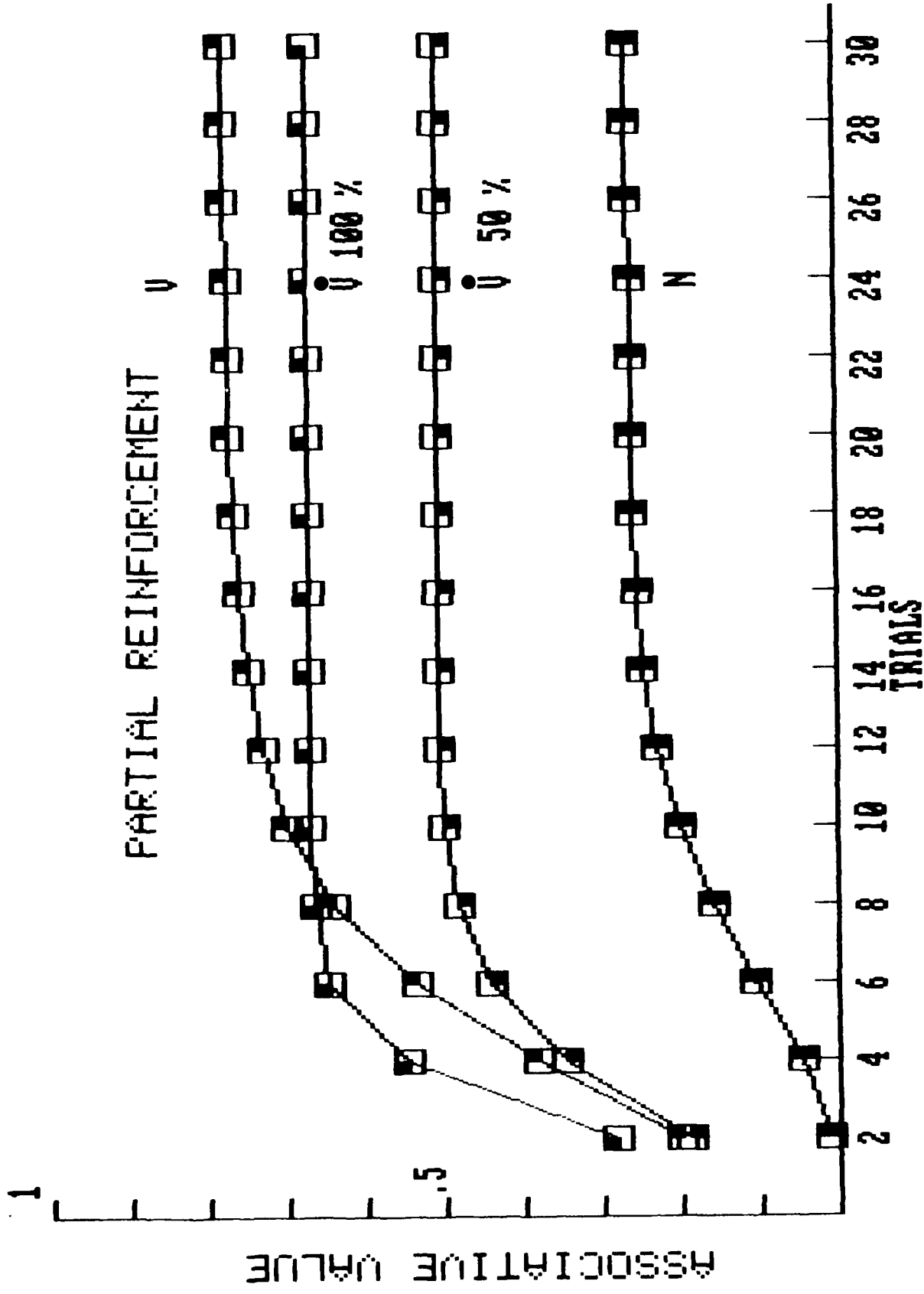**Figure 6.** Partial reinforcement with the alternative Pearce–Hall model using Equation 20. Net associative value $\dot{V}$ as a function of trials under 50% and 80% reinforcement. (1): $\gamma = 1$; (2)$\gamma = .5$.

**Figure 7.** Partial reinforcement with the alternative Pearce–Hall model using Equations 21 and 22. Net associative value $V$ as a function of trials under 50% and 80% reinforcement. (1): Equation 21 ; (2): Equation 22.
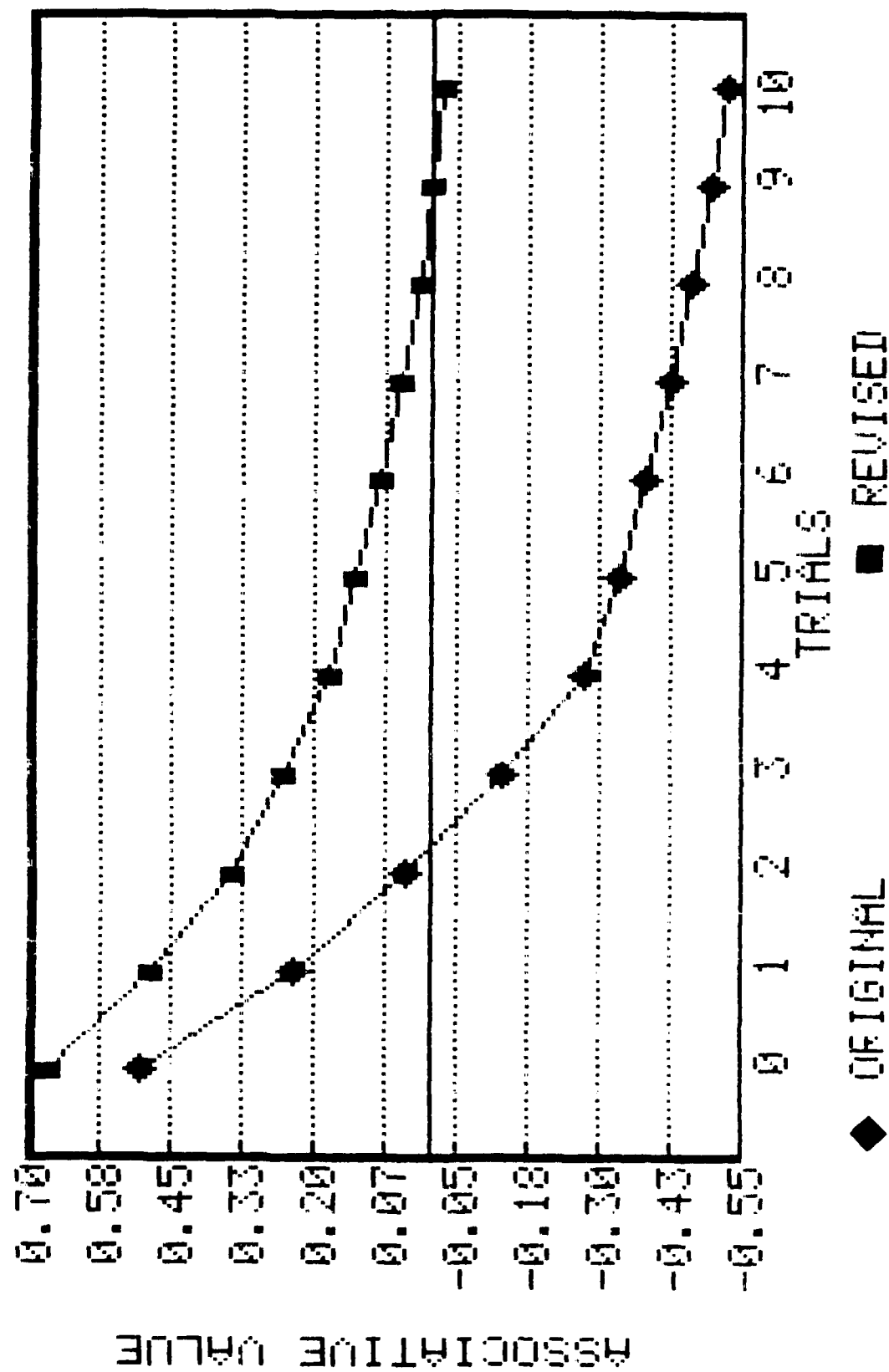
**Figure 8.** Partial reinforcement with the original and alternative Pearce–Hall models. Net associative value as a function of percentage of reinforcement: P–H, $n-1$, $\beta_E < \beta_I$, refers to original rule for computing $\alpha$ using Equation 16 with $\gamma= .5$; P–H, $n-1$, $\beta_E = \beta_I$, refers to the original rule for computing $\alpha$ using Equation 16 with $\gamma= .5$; P–H, $n$, $\beta_E = \beta_I$, refers to the alternative rule for computing $\alpha$ using Equation 20 with $\gamma = .5$; P–H$_{R-W}$ $n-1$, $\beta_E = \beta_I$, refers to the alternative rule for computing $\dot{V}$ using Equation 21 and with $\alpha$ computed using Equation 20.
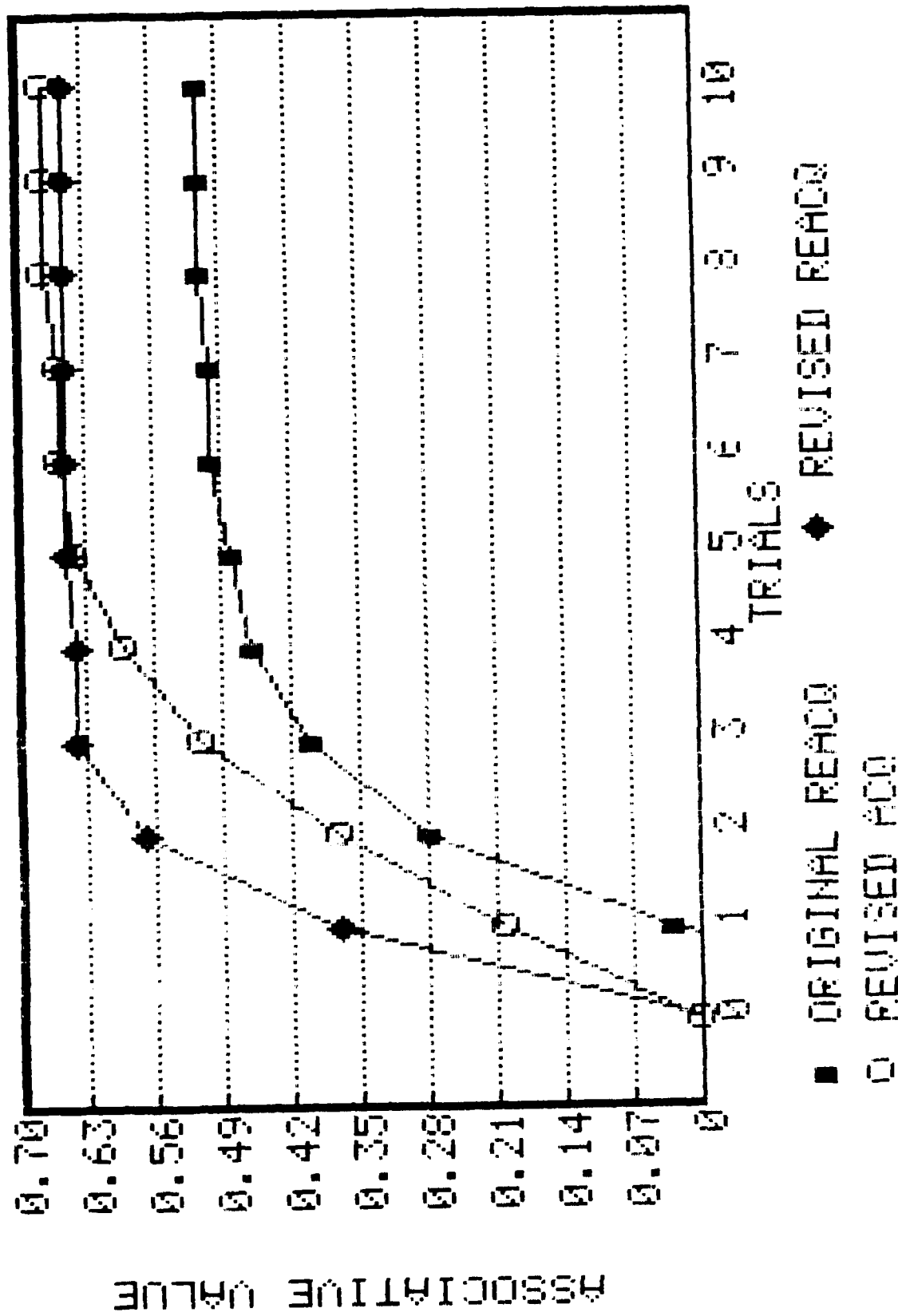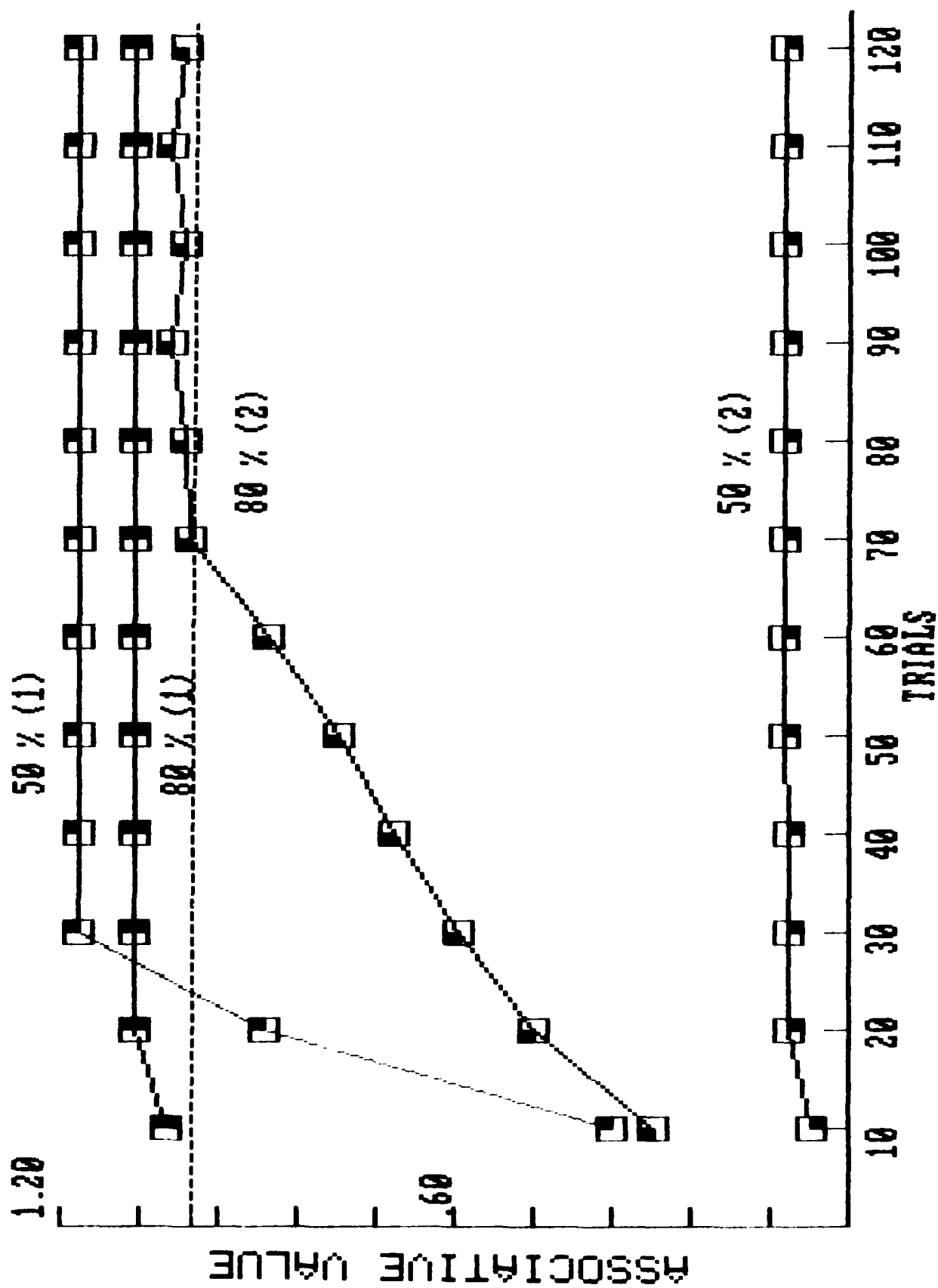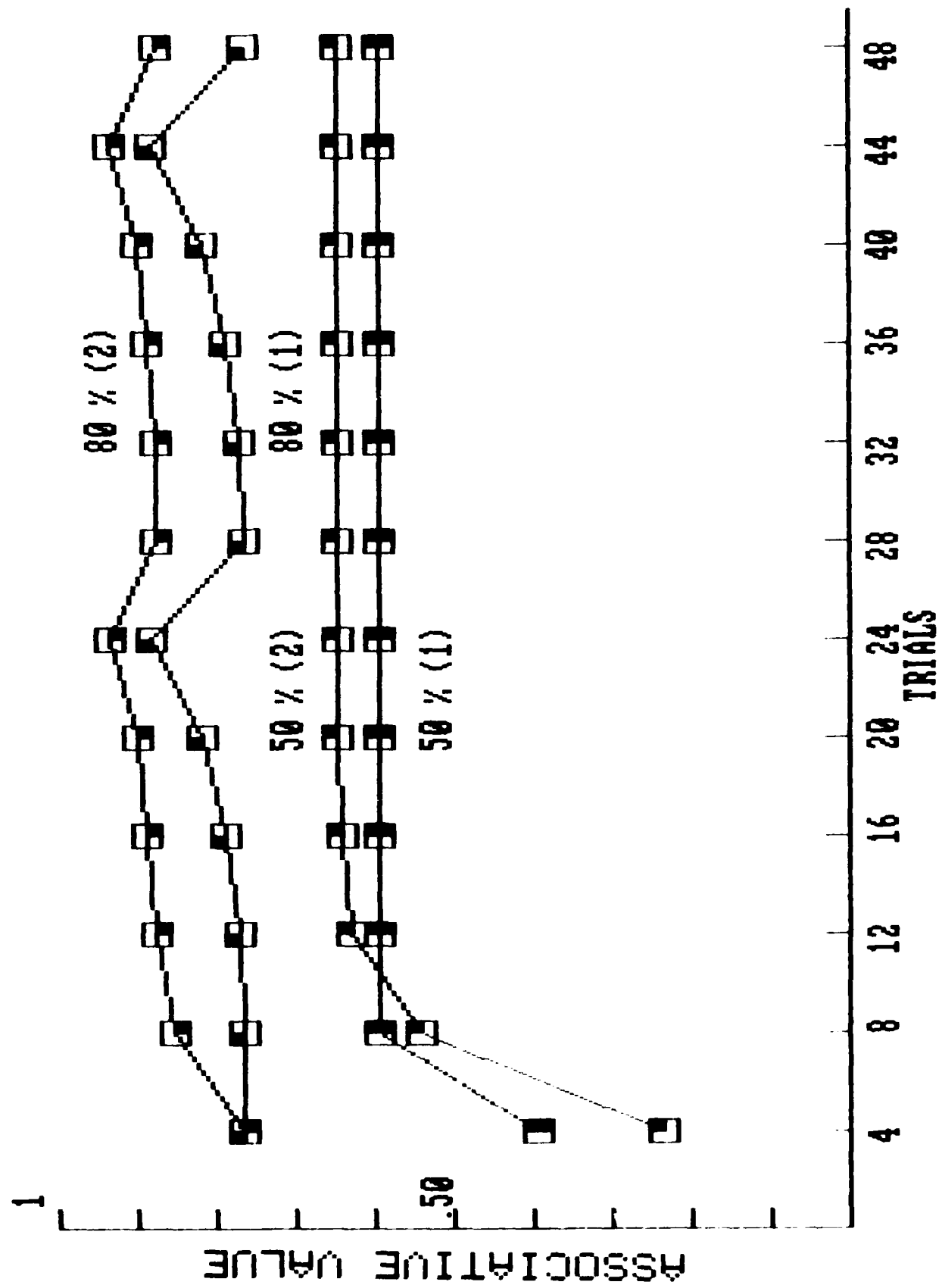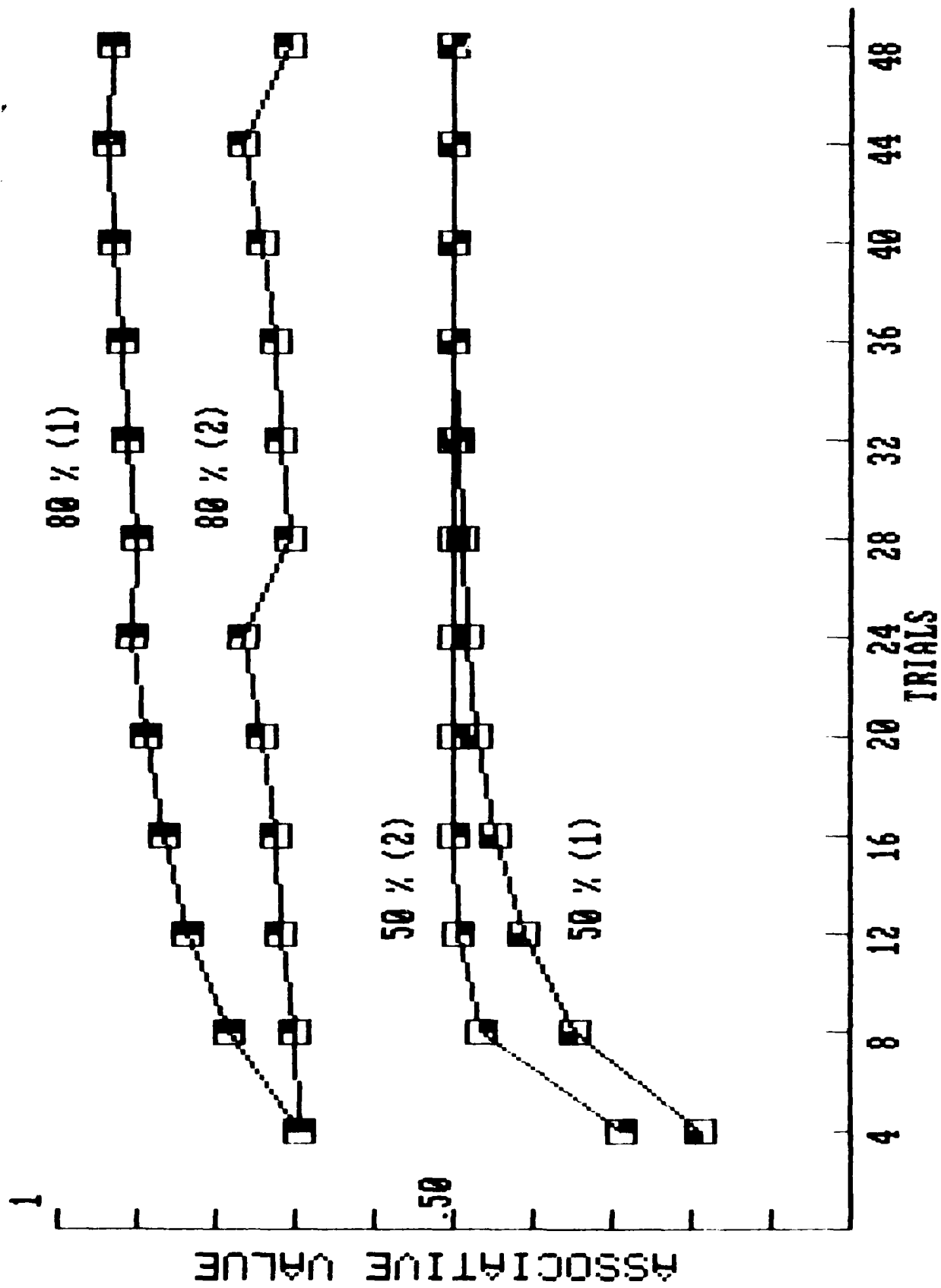
PARTIAL REINFORCEMENT

ASSOCIATIVE VALUE

TRIALS

1

PARTIAL REINFORCEMENT

ASSOCIATIVE VALUE

TRIALS

V

U̇ 100 %

U̇ 50 %

N

2

EXTINCTION

ASSOCIATIVE VALUE

◆ ORIGINAL   ■ REVISED

TRIALS

3

REACQUISITION

ASSOCIATIVE VALUE

TRIALS

0.70 0.63 0.56 0.49 0.42 0.35 0.28 0.21 0.14 0.07 0

0 1 2 3 4 5 6 7 8 9 10

■ ORIGINAL REACQ     ◆ REVISED REACQ

□ REVISED ACQ

4

5

ASSOCIATIVE VALUE

TRIALS

50 % (1)

80 % (1)

80 % (2)

50 % (2)

**ASSOCIATIVE VALUE** vs **TRIALS**

80 % (2)

80 % (1)

50 % (2)

50 % (1)

6

$\infty$.

Net Associative Value
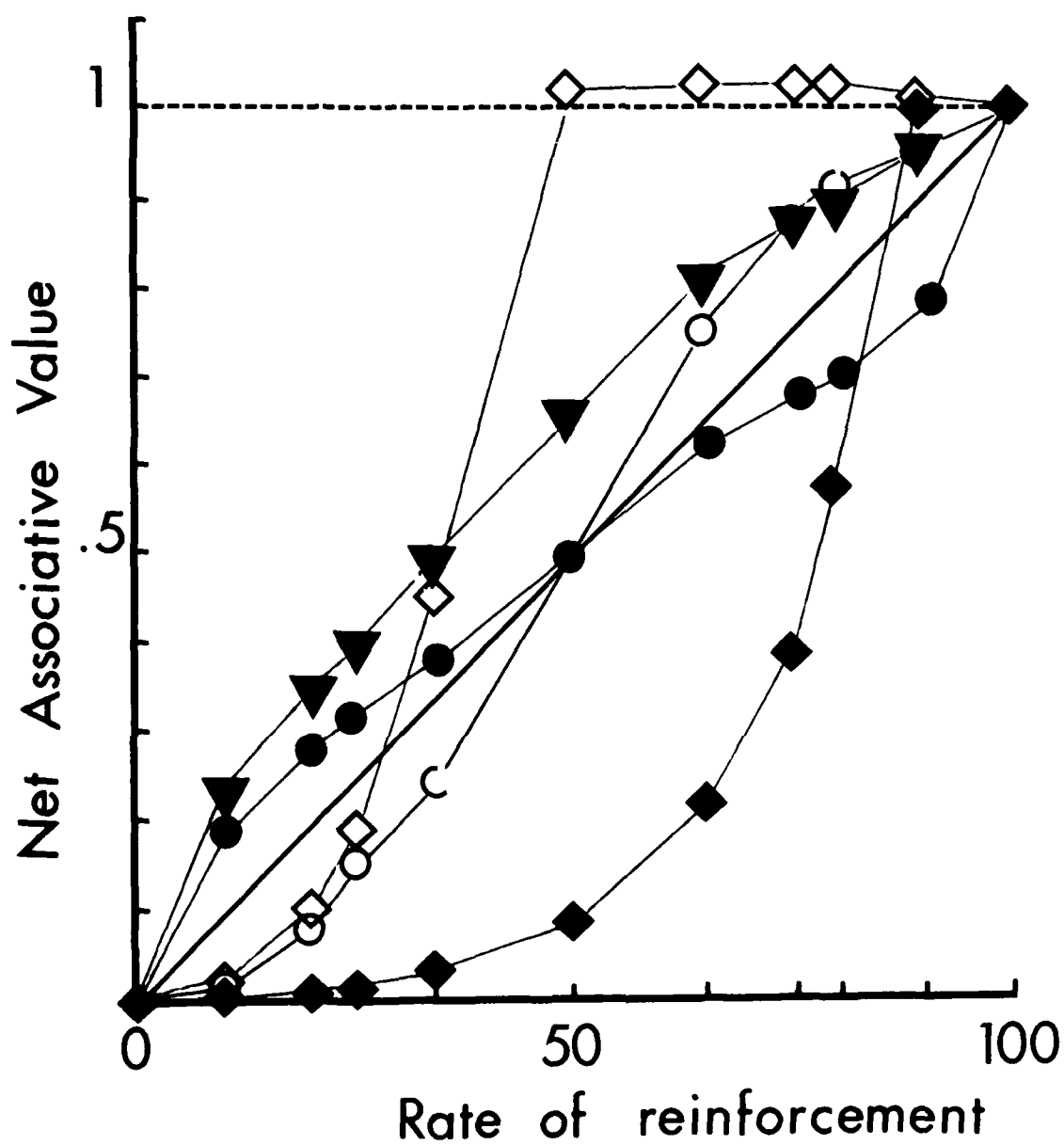
1

.5

0        50        100

Rate of reinforcement

◆ P-H, n−1, $\beta_i > \beta_e$          ○ R-W, n−1, $\beta_i = \beta_e$

◇ P-H, n−1, $\beta_i = \beta_e$          ● R-W, n, $\beta_i = \beta_e$

▼ P-H, n, $\beta_i = \beta_e$

# END

# FILMED

## MARCH, 1988

## DTIC